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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	AUG 10	Time limit for inactive STN sessions doubles to 40 minutes
NEWS	3	AUG 18	COMPENDEX indexing changed for the Corporate Source (CS) field
NEWS	4	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS	5	AUG 24	CA/CAPLUS enhanced with legal status information for U.S. patents
NEWS	6	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
NEWS	7	SEP 11	WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus
NEWS	8	OCT 21	Derwent World Patents Index Coverage of Indian and Taiwanese Content Expanded
NEWS	9	OCT 21	Derwent World Patents Index enhanced with human translated claims for Chinese Applications and Utility Models
NEWS	10	NOV 23	Addition of SCAN format to selected STN databases
NEWS	11	NOV 23	Annual Reload of IFI Databases
NEWS	12	DEC 01	FRFULL Content and Search Enhancements
NEWS	13	DEC 01	DGENE, USGENE, and PCTGEN: new percent identity feature for sorting BLAST answer sets
NEWS	14	DEC 02	Derwent World Patent Index: Japanese FI-TERM thesaurus added
NEWS	15	DEC 02	PCTGEN enhanced with patent family and legal status display data from INPADOCDB
NEWS	16	DEC 02	USGENE: Enhanced coverage of bibliographic and sequence information
NEWS	17	DEC 21	New Indicator Identifies Multiple Basic Patent Records Containing Equivalent Chemical Indexing in CA/CAPLUS
NEWS	18	JAN 12	Match STN Content and Features to Your Information Needs, Quickly and Conveniently
NEWS	19	JAN 25	Annual Reload of MEDLINE database
NEWS	20	FEB 16	STN Express Maintenance Release, Version 8.4.2, Is Now Available for Download
NEWS	21	FEB 16	Derwent World Patents Index (DWPI) Revises Indexing of Author Abstracts
NEWS	22	FEB 16	New FASTA Display Formats Added to USGENE and PCTGEN
NEWS	23	FEB 16	INPADOCDB and INPAFAMDB Enriched with New Content and Features
NEWS	24	FEB 16	INSPEC Adding Its Own IPC codes and Author's E-mail Addresses

NEWS EXPRESS FEBRUARY 15 10 CURRENT WINDOWS VERSION IS V8.4.2,
AND CURRENT DISCOVER FILE IS DATED 15 JANUARY 2010.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 10:49:58 ON 01 APR 2010

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.22	0.22

FILE 'REGISTRY' ENTERED AT 10:50:12 ON 01 APR 2010

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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 31 MAR 2010 HIGHEST RN 1215265-65-8

DICTIONARY FILE UPDATES: 31 MAR 2010 HIGHEST RN 1215265-65-8

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TSCA INFORMATION NOW CURRENT THROUGH January 8, 2010.

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REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> s galanthamine

L1 376 GALANTHAMINE

=> s lycoramine

L2 45 LYCORAMINE

=> s rivastigmine

L3 5 RIVASTIGMINE

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	16.99	17.21

FILE 'CAPLUS' ENTERED AT 10:50:38 ON 01 APR 2010
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FILE COVERS 1907 - 1 Apr 2010 VOL 152 ISS 14
FILE LAST UPDATED: 31 Mar 2010 (20100331/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2009

Caplus now includes complete International Patent Classification (IPC) reclassification data for the first quarter of 2010.

CAS Information Use Policies apply and are available at:

<http://www.cas.org/legal/infopolicy.html>

This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> s l1 or l2 or l3
      1990 L1
      137 L2
      894 L3
L4      2481 L1 OR L2 OR L3

=> s l4 and (delay?)(S)(release)
      221090 DELAY?
      582050 RELEASE
      28914 RELEASES
      599845 RELEASE
          (RELEASE OR RELEASES)
      5848 (DELAY?)(S)(RELEASE)
L5      10 L4 AND (DELAY?)(S)(RELEASE)

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6      10 DUP REM L5 (0 DUPLICATES REMOVED)

=> s l6 and ad<19981123
L7      10 S L6
          3465898 AD<19981123
          (AD<19981123)
L8      0 L7 AND AD<19981123

=> d l5 1-10 ibib abs
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L5 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2009:1401352 CAPLUS
DOCUMENT NUMBER: 151:537048
TITLE: Pharmaceutical comprising galanthamine having

INVENTOR(S): Muskulus, Frank; Paetz, Jana
 PATENT ASSIGNEE(S): Ratiopharm G.m.b.H., Germany
 SOURCE: PCT Int. Appl., 24pp.; Chemical Indexing Equivalent to 151:537025 (EP)
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: German
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2009135623	A1	20091112	WO 2009-EP3146	20090430
W:	AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
EP 2116232	A1	20091111	EP 2008-8779	20080509
R:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, AL, BA, MK, RS			

PRIORITY APPLN. INFO.: EP 2008-8779 A 20080509
 AB The invention relates to a pharmaceutical for the oral administration of the active ingredient galanthamine, comprising pellets and at least one tablet, wherein the pellets comprise an inert core and at least a coating comprising the active ingredient that releases the active ingredient in an extended manner and the tablet quickly releases the active ingredient. Thus a delayed-release pellet contained (mg): galanthamine hydrobromide 7.8; triethylcellulose 6.1; Aquacoat ECD 33; Cellets 500 37.5. An instant-release tablet included (mg): galanthamine hydrobromide 2.6; Avicel PH 102 11.4; Tablettose 80 15.4; Kollidone VA 64 1.6; Explotab 1.3; magnesium stearate 0.3. One pellet and one tablet were encapsulated in a hard gelatin capsule; dissoln. of the drug was tested.
 REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN
 ACCESSION NUMBER: 2009:1388057 CAPLUS
 DOCUMENT NUMBER: 151:537025
 TITLE: Pharmaceutical comprising galanthamine having controlled release
 INVENTOR(S): Muskulus, Frank; Paetz, Jana
 PATENT ASSIGNEE(S): Ratiopharm GmbH, Germany
 SOURCE: Eur. Pat. Appl., 13pp.; Chemical Indexing Equivalent to 151:537048 (WO)
 CODEN: EPXXDW
 DOCUMENT TYPE: Patent
 LANGUAGE: German
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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EP 2116232 A1 20091111 EP 2008-8779 20080509
 R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,
 IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI,
 SK, TR, AL, BA, MK, RS
 WO 2009135623 A1 20091112 WO 2009-EP3146 20090430
 W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ,
 CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES,
 FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE,
 KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD,
 ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH,
 PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ,
 TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
 RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,
 IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI,
 SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
 TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM,
 ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.:

EP 2008-8779

A 20080509

AB The invention relates to a pharmaceutical for the oral administration of the active ingredient galanthamine, comprising pellets and at least one tablet, wherein the pellets comprise an inert core and at least a coating comprising the active ingredient that releases the active ingredient in an extended manner and the tablet quickly releases the active ingredient. Thus a delayed-release pellet contained (mg): galanthamine hydrobromide 7.8; triethylcellulose 6.1; Aquacoat ECD 33; Cellets 500 37.5. An instant-release tablet included (mg): galanthamine hydrobromide 2.6; Avicel PH 102 11.4; Tablettose 80 15.4; Kollidone VA 64 1.6; Explotab 1.3; magnesium stearate 0.3. One pellet and one tablet were encapsulated in a hard gelatin capsule; dissoln. of the drug was tested.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2008:1338451 CAPLUS

DOCUMENT NUMBER: 149:541636

TITLE: Combination pharmaceutical compositions comprising minicapsules or minispheres of, for example, nimodipine and tacrolimus

INVENTOR(S): Coulter, Ivan

PATENT ASSIGNEE(S): Sigmoid Pharma Ltd., Ire.

SOURCE: PCT Int. Appl., 109pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008132712	A2	20081106	WO 2008-IE53	20080501
WO 2008132712	A3	20100218		
W:	AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW,			

AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
 CA 2685593 A1 20081106 CA 2008-2685593 20080501
 EP 2063875 A2 20090603 EP 2008-738144 20080501
 R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,
 IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI,
 SK, TR, AL, BA, MK, RS

PRIORITY APPLN. INFO.: US 2007-924132P P 20070501
 WO 2008-IE53 W 20080501

AB A modified release dosage product is provided, comprising a plurality of minicapsules or minispheres containing various active agents, for example, a calcium channel blocker, such as nimodipine, and/or a calcineurin inhibitor, such as tacrolimus. Uncoated minicapsules or minispheres encapsulating micronized nimodipine for immediate release and a controlled release polymer coated minicapsule or minisphere encapsulating micronized nimodipine for delayed, sustained, controlled or targeted release are described. Uncoated seamless minicapsules, the core of which comprise tacrolimus lipid-based formulation for immediate release and a controlled release polymer coated seamless minicapsule, the core of which comprises tacrolimus lipid-based formulation for delayed, sustained, controlled release or targeted release are also described. The final dosage form may be a hard gelatin capsule. Thus, nimodipine multiparticulate seamless minicapsules were produced containing nimodipine 37.5%, gelatin 56.3% and sorbitol 6.3%, and some of the minicapsules were coated with Surelease. Tacrolimus minicapsules were also produced comprising a core containing tacrolimus 3.25%, Labrafil 36.4%, olive oil 47.65%, and ethanol 12.7%, and a shell containing gelatin 90.0% and sorbitol 10.0%, and some of the minicapsules were first coated with Eudragit RS30D followed by Eudragit FS30D. The uncoated and coated nimodipine minicapsules and uncoated and coated tacrolimus minicapsules were blended into the final dosage form.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
 (1 CITINGS)

L5 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2008:973968 CAPLUS

DOCUMENT NUMBER: 149:267763

TITLE: Carbamate compounds that inhibit cholinesterase and their preparation and use in the treatment of diseases
 INVENTOR(S): Rupniak, Nadia M. J.; White, James F.; Shiosaki, Kazumi; Leander, J. David; Du, Shoucheng; Coughlin, Daniel J.

PATENT ASSIGNEE(S): Colucid Pharmaceuticals, Inc., USA

SOURCE: PCT Int. Appl., 120 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008097546	A2	20080814	WO 2008-US1516	20080204
WO 2008097546	A3	20090115		
W:	AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,			

DOCUMENT NUMBER: 145:110313
 TITLE: Pharmaceutical compositions comprising an agent with serotonin receptor modulating activity for sleep disorders
 INVENTOR(S): Rariy, Roman V.; Heffernan, Michael
 PATENT ASSIGNEE(S): Collegium Pharmaceutical, Inc., USA
 SOURCE: PCT Int. Appl., 57 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006069030	A1	20060629	WO 2005-US46049	20051220
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005319367	A1	20060629	AU 2005-319367	20051220
CA 2590802	A1	20060629	CA 2005-2590802	20051220
EP 1833467	A1	20070919	EP 2005-854713	20051220
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
JP 2008524332	T	20080710	JP 2007-548372	20051220
US 20080200508	A1	20080821	US 2007-793392	20070619
CN 101132777	A	20080227	CN 2005-80043729	20070620
IN 2007DN04915	A	20070817	IN 2007-DN4915	20070626
KR 2007087678	A	20070828	KR 2007-716730	20070720
PRIORITY APPLN. INFO.:			US 2004-637655P	P 20041220
			WO 2005-US46049	W 20051220

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Pharmaceutical compns. are provided for the pharmacol. treatment of breathing disorders and, more specifically, to compns. containing agents having serotonin receptor modulating activity for the alleviation of sleep apnea (central and obstructive) and other sleep-related breathing disorders wherein the active ingredients are released such as to extend effective blood plasma concns. across the period of sleep. For example, ondansetron immediate release tablets were prepared containing ondansetron HCl dihydrate 9.98 mg, lactose 29.14 mg, Prosolv 50 29.14 mg, Ac-Di-Sol 3.75 mg, SDS 1.5 mg, Aerosil 0.75 mg, and Mg stearate 0.75 mg. Ondansetron immediate release tablets were then coated with Eudragit L100/S100 blend to obtain delayed release tablets.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2005:258583 CAPLUS

DOCUMENT NUMBER: 142:285255

TITLE: Buccal formulations of galanthamine and derivatives and use for treating Alzheimer's disease, and the abuse of alcohol and drugs

INVENTOR(S): Asmussen, Bodo; Moormann, Joachim

PATENT ASSIGNEE(S): HF Arzneimittelforschung GmbH, Germany
 SOURCE: Ger. Offen., 9 pp.
 CODEN: GWXXBX
 DOCUMENT TYPE: Patent
 LANGUAGE: German
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10338544	A1	20050324	DE 2003-10338544	20030819
AU 2004273574	A1	20050331	AU 2004-273574	20040423
CA 2536499	A1	20050331	CA 2004-2536499	20040423
WO 2005027870	A1	20050331	WO 2004-EP4325	20040423
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1656112	A1	20060517	EP 2004-729066	20040423
EP 1656112	B1	20100210		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
JP 2007509031	T	20070412	JP 2006-523531	20040423
NZ 545560	A	20091127	NZ 2004-545560	20040423
AT 457164	T	20100215	AT 2004-729066	20040423
US 20070190117	A1	20070816	US 2006-569160	20061017
PRIORITY APPLN. INFO.:			DE 2003-10338544	A 20030819
			WO 2004-EP4325	W 20040423

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention concerns film-shaped buccal formulations of galanthamine, its salts and derivs. as central nervous system cholinergic drug or a combination of two of these drugs. The drug is embedded in polymeric matrix layers; the film can be mucus-adhesive or not adhering to the mucus. Controlled-release formulations are prepared The buccal delivery systems is used to treat Alzheimer's disease, the abuse of alc. and drugs, as antidote for neuroleptic anesthesia, and nervous system drug.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2001:152471 CAPLUS

DOCUMENT NUMBER: 134:198101

TITLE: Delayed-release pharmaceutical formulations containing acrylic polymers

INVENTOR(S): Andina, Christian; Fanning, Niall; Palmer, Fiona; Stark, Paul

PATENT ASSIGNEE(S): Elan Corporation, Plc., Ire.

SOURCE: PCT Int. Appl., 38 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001013898	A2	20010301	WO 2000-GB3309	20000829
WO 2001013898	A3	20010525		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2380333	A1	20010301	CA 2000-2380333	20000829
AU 2000067156	A	20010319	AU 2000-67156	20000829
EP 1206250	A2	20020522	EP 2000-954802	20000829
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003507416	T	20030225	JP 2001-518036	20000829
PRIORITY APPLN. INFO.:			US 1999-150880P	P 19990826
			US 1999-150891P	P 19990826
			US 1999-151221P	P 19990826
			WO 2000-GB3309	W 20000829
AB The application discloses novel pharmaceutical formulations adapted for delaying the release of a pharmaceutically active agent. A delayed-release drug formulation encapsulates the drug, which may be applied to microparticles or in tableted form, in a release-delaying coat comprising polymeric materials of predetd. swelling/permeability characteristics. In particular, acrylate and/or acrylic acid polymer blends modified with ionic groups may be used. One preferred embodiment uses a polymer of pH dependent permeability as a more permeable element of the coat. The delayed-release formulations are deployed in a single dosage form together with instant release or sustained release formulations, so that a unit dosage form, preferably an oral dosage form, can effectively administer 2 doses to a patient at different times. Thus, sustained release minitabets were formulated and encapsulated into hard gelatin capsules by using rivastigmine HTA 4.8, Methocel K100LV 14.0, Avicel PH101 15.3, Aerosil-200 0.5, and Mg stearate 0.4 mg/capsule.				
OS.CITING REF COUNT:		3	THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)	
REFERENCE COUNT:		4	THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT	
L5 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN				
ACCESSION NUMBER:		2000:880940 CAPLUS		
DOCUMENT NUMBER:		134:46786		
TITLE:		Delayed total release two pulse gastrointestinal drug delivery system		
INVENTOR(S):		Penhasi, Adel; Flashner, Moshe; Lerner, E. Itzhak		
PATENT ASSIGNEE(S):		Perio Products Ltd., Israel		
SOURCE:		PCT Int. Appl., 96 pp. CODEN: PIXXD2		
DOCUMENT TYPE:		Patent		
LANGUAGE:		English		
FAMILY ACC. NUM. COUNT:		1		
PATENT INFORMATION:				

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000074655	A2	20001214	WO 2000-US15185	20000602

WO 2000074655 A3 20010830

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 20020110593 A1 20020815 US 1999-325748 19990604

US 6632451 B2 20031014

CA 2375714 A1 20001214 CA 2000-2375714 20000602

CA 2375714 C 20081007

EP 1189601 A2 20020327 EP 2000-939503 20000602

EP 1189601 B1 20041222

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

AT 285228 T 20050115 AT 2000-939503 20000602

AU 780141 B2 20050303 AU 2000-54582 20000602

IL 146708 A 20080605 IL 2000-146708 20000602

PRIORITY APPLN. INFO.: US 1999-325748 A 19990604

WO 2000-US15185 W 20000602

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A two pulse gastrointestinal delivery system is provided. The system comprises a desired agent in combination with a swellable core material, the core being surrounded by an inner coat of a water-insol. or relatively water-insol. coating material in which particulate water-insol. material is embedded. The inner coat is addnl. surrounded by an outer coat that contains addnl. amts. of the desired agent. When the delivery device enters the gastrointestinal tract, the outer coat releases the desired agent contained therein and disintegrates, exposing the inner coat. The particulate matter in the inner coat takes up liquid, thus forming channels interconnecting the drug-containing core with the outside of the delivery device. Through these channels liquid enters the core which then swells to the point at which the inner coat is broken. When the integrity of the inner coat is destroyed, the core then disintegrates, immediately releasing all or most of the drug at a specific site. By controlling parameters in the device, such as the core material, carrier material in the coating, and particulate matter, the location of release of both pulses of the drug can be carefully controlled. The invention is also directed to a method of using the device for the treatment of disease by the release of drugs in the gastrointestinal tract in a location- and time-dependent manner. A tablet core was prepared from Ca pectinate 59, Emcocel 20, crosslinked PVP10, Na diclofenac 10, and Mg stearate 1%.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2000:314578 CAPLUS

DOCUMENT NUMBER: 132:318050

TITLE: Choline esterase inhibitors, alone or with other agents, for treating restless legs syndrome and/or periodic limb movements during sleep, and diagnostic method

INVENTOR(S): Hedner, Jan; Kraiczi, Holger

PATENT ASSIGNEE(S): Swed.

SOURCE: PCT Int. Appl., 26 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000025821	A1	20000511	WO 1999-SE1979	19991103
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1154795	A1	20011121	EP 1999-957453	19991103
EP 1154795	B1	20050817		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
AT 302025	T	20050915	AT 1999-957453	19991103
ES 2251242	T3	20060416	ES 1999-957453	19991103
PRIORITY APPLN. INFO.:			SE 1998-3760	A 19981104
			WO 1999-SE1979	W 19991103

AB A method for treating or preventing the restless legs syndrome and/or the periodic limb movements during sleep comprises administration of a choline esterase inhibitor (CEI) and, optionally, carbamazepine, clonidine, baclofen, hypnotic agent, opioid agonist, and dopaminergic agonist. Administration precedes the onset of sleep at night by from zero to three hours so as to make the CEI exert a therapeutic effect during a major portion of the sleep period. Also disclosed are corresponding pharmaceutical compns. and their use, including compns. comprising a combination of CEI with carbamazepine, clonidine, baclofen, hypnotic agent, opioid agonist, and dopaminergic agonist.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 1999:252855 CAPLUS

DOCUMENT NUMBER: 131:111271

TITLE: Further studies on Nivalin P-induced changes in muscle fiber membrane processes

AUTHOR(S): Radicheva, N.; Mileva, K.; Stoyanova, N.; Georgieva, B.

CORPORATE SOURCE: Institute of Biophysics, Bulgarian Academy of Sciences, Sofia, Bulg.

SOURCE: Methods and Findings in Experimental and Clinical Pharmacology (1999), 21(1), 5-10
CODEN: MFEPDX; ISSN: 0379-0355

PUBLISHER: Prous Science

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Nivalin P, composed of Nivalin (galanthamine hydrobromide) and Pimadin (4-aminopyridine hydrochloride), was applied extracellularly to isolated skeletal muscle fibers during prolonged activity (fatiguing) to better understand the effects of the drug on membrane ionic processes. Changes in intracellular action potential (ICAP) and twitch (Tw) parameters were monitored from treated and untreated fibers during uninterrupted activity (endurance time, ET) produced by repetitive stimulation every 200 ms for 3 min. Nivalin P-induced a shortening of the ET, drastic changes in repolarization of the ICAP corresponding to changes in neg. after-potential and falling area and an initial increase of the Tw amplitude and duration. These results suggest that Nivalin P: (i) inhibits the Na⁺,K⁺-pump due to nonspecific reduction of Na⁺ influx, stimulates the Na⁺-Ca²⁺ exchanger and inhibits K⁺ conductance: (ii) increases Ca²⁺ release and delays Ca²⁺ uptake under sufficient depolarization. It was concluded that fatigue develops faster

in the presence of Nivalin P.
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD
(3 CITINGS)
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file medline embase biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	39.93	57.14
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-8.50	-8.50

FILE 'MEDLINE' ENTERED AT 10:52:46 ON 01 APR 2010

FILE 'EMBASE' ENTERED AT 10:52:46 ON 01 APR 2010
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FILE 'BIOSIS' ENTERED AT 10:52:46 ON 01 APR 2010
Copyright (c) 2010 The Thomson Corporation

=> d his

(FILE 'HOME' ENTERED AT 10:49:58 ON 01 APR 2010)

FILE 'REGISTRY' ENTERED AT 10:50:12 ON 01 APR 2010

L1 376 S GALANTHAMINE
L2 45 S LYCORAMINE
L3 5 S RIVASTIGMINE

FILE 'CAPLUS' ENTERED AT 10:50:38 ON 01 APR 2010

L4 2481 S L1 OR L2 OR L3
L5 10 S L4 AND (DELAY?) (S) (RELEASE)
L6 10 DUP REM L5 (0 DUPLICATES REMOVED)
L7 10 S L6
L8 0 S L6 AND AD<19981123

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:46 ON 01 APR 2010

=> s acetylcholinesterase(S)(delay?)(S)(release)
L9 4 ACETYLCHOLINESTERASE(S)(DELAY?)(S)(RELEASE)

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 2 DUP REM L9 (2 DUPLICATES REMOVED)

=> d l10 1-2 ibib abs

L10 ANSWER 1 OF 2 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN DUPLICATE 1

ACCESSION NUMBER: 1993112492 EMBASE

TITLE: Prejunctional action of neostigmine on mouse neuromuscular
preparations.

AUTHOR: Braga, M.F.M.; Rowan, E.G.; Harvey, A.L.; Bowman, W.C.
(correspondence)

CORPORATE SOURCE: Dept Physiology and Pharmacology, Strathclyde Inst for Drug
Research, University of Strathclyde, Glasgow G1 1XW, United
Kingdom.

SOURCE: British Journal of Anaesthesia, (1993) Vol. 70, No. 4, pp.

405-410.
ISSN: 0007-0912 CODEN: BJANAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 024 Anesthesiology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 May 1993
Last Updated on STN: 16 May 1993

AB We have studied the effects of neostigmine on the mouse diaphragm and triangularis sterni isolated nerve-muscle preparations. Mechanical responses of the muscle, end-plate potentials and miniature end-plate potentials, and extracellularly recorded nerve ending currents were recorded. In the mouse diaphragm nerve-muscle preparations, neostigmine 1 $\mu\text{mol litre}^{-1}$ continued to produce some antagonism of tubocurarine-induced block after cholinesterase had been inactivated completely by diisopropyl fluorophosphate 22 $\mu\text{mol litre}^{-1}$. In the mouse triangularis sterni preparation, neostigmine 0.1-1 $\mu\text{mol litre}^{-1}$ increased the quantal content of the end-plate potential in a concentration-dependent manner. This effect appeared to be sufficient to account for the cholinesterase-independent antagonistic action to tubocurarine under the conditions of the experiments. Neostigmine 1-100 $\mu\text{mol litre}^{-1}$ depressed the amplitude of the K^+ currents of the perineural waveforms in a concentration-dependent manner, and this may account for its ability to increase the quantal content of the end-plate potential. Although inhibition of acetylcholinesterase is the main mechanism of action of neostigmine, the drug also exerts an additional direct action on motor nerve endings to block the delayed rectifier K^+ channels and enhance transmitter release. This effect occurred at clinically relevant concentrations of neostigmine. Physostigmine and pyridostigmine did not possess this additional action.

L10 ANSWER 2 OF 2 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN DUPLICATE 2

ACCESSION NUMBER: 1981149516 EMBASE
TITLE: The pharmacology of pumiliotoxin-B. I. Interaction with calcium sites in the sarcoplasmic reticulum of skeletal muscle.
AUTHOR: Albuquerque, E.X.; Warnick, J.E.; Maleque, M.A.; et. al.
CORPORATE SOURCE: Dept. Pharmacol. Exp. Therapeut., Sch. Med., Univ. Maryland, Baltimore, Md. 21201, United States.
SOURCE: Molecular Pharmacology, (1981) Vol. 19, No. 3, pp. 411-424.
ISSN: 0026-895X CODEN: MOPMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB The actions of pumiliotoxin-B (PTX-B), a novel indolizidine alkaloid from the skin of the Panamanian frog, *Dendrobates pumilio*, have been studied in skeletal muscle of rat, frog, and crayfish with electrophysiological and biochemical techniques. PTX-B reversibly potentiates and prolongs the direct elicited muscle twitch in rat and frog skeletal muscle up to 12-fold in a concentration- and frequency-dependent manner, the potentiation being greater at the lower frequencies of stimulation.

Responses of the muscle to tetanic stimulation in the presence of PTX-B are potentiated more at 10 and 20 Hz than at 50 and 100 Hz; tetanic fusion occurs earlier, and an aftercontraction is present when tetanic stimulation occurs in the presence of PTX-B. The twitch/tetanus ratio at 100 Hz is increased in the presence of PTX-B from 0.3 to more than 1.1 as a result of the increase in twitch amplitude. These effects on frog skeletal muscle are seen in the absence of any effect of PTX-B on spontaneous and evoked transmitter release, acetylcholinesterase activity, muscle action potential, delayed rectification, and cable properties of the muscle fiber. In the absence of external calcium, PTX-B prolongs but does not potentiate the twitch, while methoxyverapamil and dantrolene only partially suppress the actions of PTX-B. In crayfish skeletal muscle, PTX-B increases the rate of rise of the 'calcium-dependent' action potential and shortens its duration. Biochemical studies reveal that PTX-B inhibits calcium-dependent adenosine triphosphatase from sarcoplasmic reticulum preparations of both frog and rat skeletal muscles in a concentration- and calcium-dependent manner. We suggest that PTX-B potentiates and prolongs the muscle twitch by (a) facilitating the release of calcium from storage sites within the sarcoplasmic reticulum, (b) mobilizing calcium from extracellular sites, and (c) blocking the reuptake of calcium by calcium-dependent adenosine triphosphatase.

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=> s delay?(A)release or program?(a)release
L11      9349 DELAY?(A) RELEASE OR PROGRAM?(A) RELEASE
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=> s l11 and acetylcholinesterase(S)inhibitor
L12      1 L11 AND ACETYLCHOLINESTERASE(S) INHIBITOR
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=> d l12 ibib abs
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L12 ANSWER 1 OF 1 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
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ACCESSION NUMBER: 0002501967 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: Growth hormone response to overnight growth
hormone-releasing hormone infusion and oral pyridostigmine
in children with short stature..
AUTHOR: Ross, R.J. (correspondence); Savage, M.O.; Kirk, J.M.;
Besser, G.M.
CORPORATE SOURCE: Department of Endocrinology, St Bartholomew's Hospital,
London, UK..
SOURCE: Acta paediatrica Scandinavica. Supplement, (1989) Vol. 349,
pp. 114-116; discussion 123-124.
ISSN: 0300-8843
COUNTRY: Sweden
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010
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AB The development of a long-acting or depot preparation of growth
hormone-releasing hormone (GHRH) may have many advantages over
conventional treatment (with GH) of GH-deficient children.
Pyridostigmine, an acetylcholinesterase inhibitor, has
been shown to augment basal GH secretion and the GH response to GHRH in
short children. It may thus provide adjuvant therapy to depot GHRH. The
GH response to a nocturnal subcutaneous infusion of GHRH (1-29)NH2 in
doses of 5 and 10 micrograms/kg/hour was investigated in five short,
slowly growing children. The effect of oral pyridostigmine 60 mg on
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nocturnal GH secretion and the GH response to a nocturnal infusion was also examined. The subcutaneous infusion of GHRH augmented pulsatile GH release in all five children. There was a dose-related response to subcutaneous GHRH for the GH area under the curve and mean GH pulse amplitude, but no change in the number of pulses. There was a significant rise in the mean baseline GH concentration during the GHRH infusion compared with placebo. Pyridostigmine had no effect on either basal or stimulated GH secretion.

=> s l11 and cholinergic

L13 59 L11 AND CHOLINERGIC

=> dup rem l13

PROCESSING COMPLETED FOR L13

L14 54 DUP REM L13 (5 DUPLICATES REMOVED)

=> s l14 and pd<19981123

2 FILES SEARCHED...

L15 31 L14 AND PD<19981123

=> s l15 and (galanthamine or lycoramine or rivastigmine)

L16 0 L15 AND (GALANTHAMINE OR LYCORAMINE OR RIVASTIGMINE)

=> d l15 1-31 ibib abs

L15 ANSWER 1 OF 31 MEDLINE on STN

ACCESSION NUMBER: 1996407511 MEDLINE

DOCUMENT NUMBER: PubMed ID: 8811564

TITLE: Neuropathy and vasculopathy in colonic strictures from children with cystic fibrosis.

AUTHOR: Collins M H; Azzarelli B; West K W; Chong S K; Maguiness K M; Stevens J C

CORPORATE SOURCE: Division of Pediatric Pathology, Indiana University School of Medicine, Riley Hospital for Children, Indianapolis, USA.

SOURCE: Journal of pediatric surgery, (1996 Jul) Vol. 31, No. 7, pp. 945-50.

Journal code: 0052631. ISSN: 0022-3468. L-ISSN: 0022-3468.

PUB. COUNTRY: United States

DOCUMENT TYPE: (COMPARATIVE STUDY)

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199612

ENTRY DATE: Entered STN: 28 Jan 1997

Last Updated on STN: 28 Jan 1997

Entered Medline: 4 Dec 1996

AB Colonic strictures are rare in patients who have cystic fibrosis, but recently have developed in those who have been treated with delayed-release high-dose pancreatic enzyme supplements. Colonic strictures from eight such pediatric patients showed neural abnormalities consisting of ganglion cell hyperplasia and ectopia, and intermyenteric plexus hyperplasia. Cholinergic and adrenergic stains of mucosal nerve fibers were more prominent in histological sections of the cystic fibrosis strictures than in sections from colons of children without cystic fibrosis. The mean grade of staining with acetylcholinesterase in the lamina propria of the strictured cystic fibrosis colons was 2.38 +/- 1.25, compared with .93 +/- .93 (P < .055) in bowels from children without cystic fibrosis. The mean grade for tyrosine hydroxylase staining in the lamina propria was 2 +/- .97 in the strictures and was .79 +/- .81 (P < .05) in the bowels of children who did not have

cystic fibrosis. Vasoactive intestinal peptide staining in bowels from children with cystic fibrosis with and without stricture did not differ significantly from that of children without cystic fibrosis. Vasculopathy consisting of fibrointimal hyperplasia in submucosal veins and mesenteric arteries was found only in colonic strictures owing to cystic fibrosis. Colonic strictures in patients with cystic fibrosis who received high-dose pancreatic enzyme supplements contain ganglion cell abnormalities, and mucosal cholinergic and adrenergic activity may be increased in these strictures. The stricture vasculopathy may be drug-related and/or related to increased catecholamine activity.

L15 ANSWER 2 OF 31 MEDLINE on STN
ACCESSION NUMBER: 1979023020 MEDLINE
DOCUMENT NUMBER: PubMed ID: 699016
TITLE: Catharanthine: a novel stimulator of pancreatic enzyme release.
AUTHOR: Williams J A
SOURCE: Cell and tissue research, (1978 Sep 5) Vol. 192, No. 2, pp. 277-84.
Journal code: 0417625. ISSN: 0302-766X. L-ISSN: 0302-766X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 20 Dec 1978

AB The plant alkaloid, catharanthine, was shown to stimulate release of amylase from pancreatic fragments and to cause extensive degranulation of pancreatic acinar cells with accumulation of membrane material in the Golgi region. The extent and time course of maximal catharanthine stimulation was comparable to that induced by the cholinergic analog bethanechol. Antimycin inhibited the action of catharanthine while atropine did not. Removal of Ca²⁺ from the incubation medium inhibited amylase release induced by catharanthine but did not affect release induced by bethanechol. Catharanthine induced a delayed release of ⁴⁵Ca²⁺ from prelabeled pancreatic fragments as compared to bethanechol. It is suggested therefore that catharanthine activates the physiological pathway controlling amylase release by causing a rise in cytoplasmic Ca²⁺ but the mechanism by which this occurs is different from that caused by physiological secretagogues.

L15 ANSWER 3 OF 31 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1995022538 EMBASE
TITLE: Pharmacokinetic considerations in gastrointestinal motor disorders.
AUTHOR: Hebbard, G.S.; Sun, W.M.; Bochner, F.; Horowitz, M., Prof. (correspondence)
CORPORATE SOURCE: Department of Medicine, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000, Australia.
SOURCE: Clinical Pharmacokinetics, (1995) Vol. 28, No. 1, pp. 41-66.
ISSN: 0312-5963 CODEN: CPKNDH
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Feb 1995
Last Updated on STN: 1 Feb 1995

AB Although it has been recognised that alterations in gastrointestinal motility, whether induced by physiological or pathological processes, have significant effects on the pharmacokinetics of orally administered drugs, this subject has received inappropriately little attention. Studies relating to this topic have focused on healthy volunteers and animals and have largely been confined to the effects of single drug doses. There is limited information about the effects of disease on pharmacokinetics under steady-state conditions. Changes in gastrointestinal motility may affect the pharmacokinetics of orally administered drugs by altering the rate of delivery, bioavailability or mucosal absorption of the drug. In general the rate of absorption and time taken to achieve maximal plasma concentrations for well absorbed drugs may be modified by changes in gastrointestinal motility, but overall bioavailability is not usually affected. In these cases the therapeutic and clinical effects of the alteration in pharmacokinetics will, therefore, depend on which parameters are important for the action of the drug. For poorly absorbed drugs both the rate of absorption and bioavailability are likely to be altered by changes in gastrointestinal motility. However, the complex effects of food and disease, as well as the properties and formulation of any drug (solubility, ease of dispersion, delayed release formulation) often make the prediction of the magnitude, or even the direction, of any effect difficult to predict. Drugs with direct effects on gastrointestinal motility may influence their own patterns of absorption. In patients with gastrointestinal motility disorders, drugs administered in a controlled release formulation, or those with poor bioavailability, are most likely to have a poorly predictable therapeutic effect. Care should be taken to ensure that the formulation of the drug, its timing of administration in relation to meals and the use of coadministered drugs optimise, or at least ensure consistent absorption.

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ACCESSION NUMBER: 1987225205 EMBASE
TITLE: Acute tabun toxicity; biochemical and histochemical consequences in brain and skeletal muscles of rat.
AUTHOR: Gupta, R.C.; Patterson, G.T.; Dettbarn, W.-D.
CORPORATE SOURCE: Department of Pharmacology, School of Medicine, Vanderbilt University, Nashville, TN 37232, United States.
SOURCE: Toxicology, (1987) Vol. 46, No. 3, pp. 329-341.
ISSN: 0300-483X CODEN: TXCYAC
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
052 Toxicology
008 Neurology and Neurosurgery
LANGUAGE: English

AB Male Sprague-Dawley rats injected s.c. with an acute non-lethal dose (200 µg/kg) of ethyl N,N-dimethylphosphoramidocyanidate (tabun) showed onset of hypercholinergic activity within 10-15 min. The maximal severity of toxicity signs was evident within 0.5-1 h and persisted for 6 h. Except for mild tremors no overt toxicity signs were evident after 24 h. Within 1 h a dramatic decline of acetylcholinesterase (AChE) activity occurred in all the brain structures (< 3%) and skeletal muscles (< 10% in soleus and hemi-diaphragm; and 32% in extensor digitorum longus (EDL)). No significant recovery was seen up to 48-72 h. Within 7 days rats became free of toxicity signs and AChE activity had recovered to about 40% in brain structures (except cortex, 14%) and 65-70% in skeletal muscles. Within 1 h the 16 S molecular form of AChE located at the neuromuscular junction was most severely inhibited in soleus, followed by hemi-diaphragm

and least in the EDL, and had fully recovered in all the muscles when examined after day 7. Muscle fiber necrosis developed within 1-3 h in soleus and hemi-diaphragm and after a delay of 24 h in EDL. The highest number of necrotic lesions in all muscles was seen at 72 h with the hemi-diaphragm maximally affected and EDL the least. To determine detoxification of tabun by non-specific binding, the activity of butyrylcholinesterase (BuChE) and carboxylesterase (CarbE) was measured. The inhibition and recovery pattern of BuChE activity was quite similar to that of AChE, except that the rate of recovery was more rapid. Within 1 h the remaining activity of CarbE was 10% in plasma, about 30% in brain structures, and 79% in liver; recovery was complete within 7 days. The inhibition of BuChE and CarbE can serve as a protective mechanism against tabun toxicity by reducing the amount available for AChE inhibition. The prolonged AChE inhibition in muscle and brain may indicate storage of tabun and delayed release from non-enzymic sites. Since tabun is a cyanophosphorus compound, the toxic effects from the released cyanide (CN) could be another reason for the delayed recovery after tabun.

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ACCESSION NUMBER: 0009443223 EMBASE
 COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
 TITLE: Long acting inhaled beta 2 agonists and inhaled anticholinergic drugs: benefits and side effects in childhood asthma..
 AUTHOR: Naspitz, C.K. (correspondence)
 CORPORATE SOURCE: Department of Pediatrics, Escola Paulista de Medicina, Sao Paulo, Brazil..
 SOURCE: Pediatric pulmonology. Supplement, (1997) Vol. 16, pp. 96-97.
 Refs: 10
 ISSN: 1054-187X
 COUNTRY: United States
 DOCUMENT TYPE: Journal; General Review; (Review)
 FILE SEGMENT: MEDLINE
 LANGUAGE: English
 ENTRY DATE: Entered STN: Mar 2010
 Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0008936515 EMBASE
 COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
 TITLE: Pilot study of controlled-release pilocarpine in normal subjects..
 AUTHOR: Lockhart, P.B. (correspondence); Fox, P.C.; Gentry, A.C.; Acharya, R.; Norton, H.J.
 CORPORATE SOURCE: Department of Dentistry, Carolinas Medical Center, Charlotte, NC 28232-2861, USA..
 SOURCE: Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics, (Nov 1996) Vol. 82, No. 5, pp. 517-524.
 ISSN: 1079-2104
 COUNTRY: United States
 DOCUMENT TYPE: Journal; Article
 FILE SEGMENT: MEDLINE
 LANGUAGE: English
 ENTRY DATE: Entered STN: Mar 2010
 Last Updated on STN: Mar 2010

AB OBJECTIVES: Current systemic treatments with sialogogues for patients with xerostomia are limited because of minimal efficacy, short duration of activity, or problems with side effects. The purpose of this pilot study was an initial assessment of safety, efficacy, duration of action, multiple dose tolerance, and side effects of a controlled-release formulation of pilocarpine hydrochloride. STUDY DESIGN: Eight healthy hospitalized subjects were given 15 mg of a controlled-release pilocarpine formulation every 12 hours for three doses. Saliva and blood samples were collected at assigned intervals. Repeated measures analysis and paired t tests were used for statistical analyses. RESULTS: A significant ($p < 0.05$) increase in both parotid and whole saliva output followed all three doses beginning within 1 hour of dosing and lasting over 10 hours. Mean plasma pilocarpine concentration reached a maximum of 8.2 ng/ml at approximately 1 hour after the first dose, 11.5 ng/ml after the third dose, and declined to near baseline (0.06 ng/ml) 24 hours after the final dose. None of the participants showed evidence of adverse effects including complaints of sweating or gastrointestinal discomfort. CONCLUSIONS: A controlled-release formulation of pilocarpine may overcome the therapeutic weaknesses of current pilocarpine preparations by prolonging salivary secretion and reducing undesirable side effects.

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ACCESSION NUMBER: 0008935855 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: Medication-induced adynamic ileus..
AUTHOR: Beall, D.P. (correspondence); Hofmann, L.V.
CORPORATE SOURCE: Department of Radiology and Radiological Science, Johns Hopkins Hospital, Baltimore, Maryland, USA..
SOURCE: Maryland medical journal (Baltimore, Md. : 1985), (May 1996) Vol. 45, No. 5, pp. 415-416.
ISSN: 0886-0572
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0007101891 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: [New method of medicinal treatment of cardiospasm].
Edin nov metod za medikamentozno lechenie na kardiospazma..
AUTHOR: Tikhlov, K. (correspondence)
SOURCE: Vutreshni bolesti, (1982) Vol. 21, No. 2, pp. 60-65.
ISSN: 0506-2772
COUNTRY: Bulgaria
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: Bulgarian
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0005920409 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of

this record.

TITLE: [Treatment of drug-induced parkinsonism-like states with a drug used in the therapy of parkinsonism (methixene HCl)]. Die Behandlung des arzneimittelbedingten Parkinsonoids mit einem Antiparkinsonikum in Retardform (Methixen-Hydrochlorid).

AUTHOR: Munnich, J.E. (correspondence)

SOURCE: Die Medizinische Welt, (1 Oct 1966) Vol. 40, pp. 2132-2135.
ISSN: 0025-8512

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: MEDLINE

LANGUAGE: German

ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0005634393 EMBASE

COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.

TITLE: A long-acting phenothiazine as a possible agent to control deviant sexual behavior..

AUTHOR: Bartholomew, A.A. (correspondence)

SOURCE: The American journal of psychiatry, (Jan 1968) Vol. 124, No. 7, pp. 917-923.
ISSN: 0002-953X

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: MEDLINE

LANGUAGE: English

ENTRY DATE: Entered STN: Mar 2010
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ACCESSION NUMBER: 0005495333 EMBASE

COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.

TITLE: [Treatment of gastric disorders with delayed-action Tralene]. Zur Behandlung von Magenbeschwerden mit Tralene retard..

AUTHOR: Zurbonsen, K. (correspondence); Lohmann, C.; Ecklebe, G.

SOURCE: Therapie der Gegenwart, (Dec 1970) Vol. 109, No. 12, pp. 1774-1776.
ISSN: 0040-5965

COUNTRY: Germany

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: MEDLINE

LANGUAGE: German

ENTRY DATE: Entered STN: Mar 2010
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ACCESSION NUMBER: 0005483689 EMBASE

COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.

TITLE: [Clinical trials in the therapy of peptic ulcer. 3. Ambulatory therapy of uncomplicated duodenal ulcer with diverse anticholinergic agents].

Klinische Versuche zur Therapie des Ulcus pepticum. 3.
Ambulante Therapie des unkomplizierten Ulcus duodeni mit
verschiedenen Anticholinergika..
AUTHOR: Gutz, H.J. (correspondence); Berndt, H.; Wolff, G.
SOURCE: Das Deutsche Gesundheitswesen, (25 Mar 1970) Vol.
25, No. 12, pp. 533-536.
ISSN: 0012-0219
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: German
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0005476355 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: Long-acting drug treatment in overall psychiatric
management..
AUTHOR: Capstick, N. (correspondence)
SOURCE: Diseases of the nervous system, (Sep 1970) Vol.
31, pp. Suppl:15-17.
ISSN: 0012-3714
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0005476354 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: Long-acting, antipsychotic agents and extrapyramidal side
effects..
AUTHOR: Simpson, G.M. (correspondence)
SOURCE: Diseases of the nervous system, (Sep 1970) Vol.
31, pp. Suppl:12-14.
ISSN: 0012-3714
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0005112021 EMBASE
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this record.
TITLE: [Enuresis].
L'enuresie..
AUTHOR: Richard, M. (correspondence)
SOURCE: L'union medicale du Canada, (Sep 1971) Vol. 100,
No. 9, pp. 1814-1817.
ISSN: 0041-6959
COUNTRY: Canada

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: French
ENTRY DATE: Entered STN: Mar 2010
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ACCESSION NUMBER: 0004947805 EMBASE
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TITLE: A quantitative study of neuroleptic-induced extrapyramidal symptoms and their response to dexetimide, a potent and long-acting antiparkinsonian agent..
AUTHOR: Dom, R. (correspondence); Van Lommel, R.; Baro, F.
SOURCE: Acta psychiatrica Scandinavica, (1971) Vol. 47, No. 4, pp. 399-410.
ISSN: 0001-690X
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004916211 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: A controlled trial of glycopyrronium and l-hyoscyamine in the long-term treatment of duodenal ulcer..
AUTHOR: Kaye, M.D. (correspondence); Rhodes, J.; Beck, P.; Sweetnam, P.M.; Davies, G.T.; Evans, K.T.
SOURCE: Gut, (Jul 1970) Vol. 11, No. 7, pp. 559-566.
ISSN: 0017-5749
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004840822 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: [Oral anticholinergic agents with depot effects].
Orale antikolinergika med depotvirkning.
AUTHOR: Haffner, J. (correspondence)
SOURCE: Tidsskrift for den Norske lægeforening, (Apr 1974) Vol. 94, No. 10, pp. 660.
ISSN: 0029-2001
COUNTRY: Norway
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: Norwegian
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004600237 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: [Quality of results obtained in digestive pathology by
acting upon motor, secretory and hormonal phenomena at the
same time].
Qualite des resultats obtenus en pathologie digestive en
agissant a la fois sur les phenomenes moteurs, secretoires
et hormonaux.
AUTHOR: Darnis, F. (correspondence); Streichenberger, G.
SOURCE: Therapeutique (La Semaine des hopitaux), (Dec 1973
) Vol. 49, No. 10, pp. 721-724.
ISSN: 0040-5922
COUNTRY: France
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: French
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004400085 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: Poisoning from delayed release
tablets..
AUTHOR: Meadow, S.R. (correspondence)
SOURCE: British medical journal, (19 Feb 1972) Vol. 1,
No. 5798, pp. 512.
ISSN: 0007-1447
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004388539 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: The effect of sustained release hexocyclium tablets on
gastric acid secretion..
AUTHOR: Alp, M.H. (correspondence); Grant, A.K.
SOURCE: The Medical journal of Australia, (1 Mar 1969)
Vol. 1, No. 9, pp. 447-449.
ISSN: 0025-729X
COUNTRY: Australia
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0004179682 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.

TITLE: Oral medications in nasal decongestion. A study among industrial workers..
AUTHOR: Ashe, G.J. (correspondence)
SOURCE: IMS, Industrial medicine and surgery, (Mar 1968) Vol. 37, No. 3, pp. 212-214.
ISSN: 0019-8536
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0003574699 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: Sustained-release anticholinergics in dystonia: case report..
AUTHOR: Samie, M.R. (correspondence)
SOURCE: Neurology, (May 1987) Vol. 37, No. 5, pp. 885-886.
ISSN: 0028-3878
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
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ACCESSION NUMBER: 0002890774 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: Progress of therapeutic agents for asthma--1) Progress of anti-asthmatic agents.
AUTHOR: Kawamura, H. (correspondence); Nagano, H.
SOURCE: Nippon rinsho. Japanese journal of clinical medicine, (Aug 1987) Vol. 45, No. 8, pp. 1830-1836.
ISSN: 0047-1852
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: Japanese
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0001981633 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: [Treatment of patients with chronic obstructive bronchitis and pulmonary hypertension during ambulatory care].
Lechenie bol'nykh khronicheskim obstruktiivnym bronkhitom s legochnoi gipertenziei na dispansernom etape..
AUTHOR: Paleev, N.R. (correspondence); Tsar'kova, L.N.; Chereiskaia, N.K.; Baklykova, S.N.
SOURCE: Sovetskaia meditsina, (1990) No. 11, pp. 66-69.
ISSN: 0038-5077

COUNTRY: Russian Federation
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: Russian
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0001425260 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: [Physical exercise tolerance in chronic obstructive emphysematous bronchitis and coronary heart disease under antiobstructive therapy].
Körperliche Belastbarkeit bei chronisch-obstruktiver Emphysebronchitis und koronarer Herzkrankheit unter antiobstruktiver Therapie..
AUTHOR: Hurter, T. (correspondence); Ochs, J.G.; Schmitz, E.; Hartmann, M.; Alex, C.; Sigmund, M.; Hanrath, P.
CORPORATE SOURCE: Medizinische Klinik I, Technische Hochschule Aachen..
SOURCE: Deutsche medizinische Wochenschrift (1946), (23 Oct 1992) Vol. 117, No. 43, pp. 1623-1629.
ISSN: 0012-0472
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: German
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

AB 19 consecutive patients (18 men, one woman, mean age 61.4 [49-73]years) with chronic obstructive airways disease (bronchitis and emphysema) together with angiographically confirmed coronary heart disease were studied to investigate their cardiopulmonary exercise tolerance and the effects of bronchodilators on their myocardial ischaemia. Because they were receiving drug therapy for angina or because they had previously undergone aortocoronary bypass operation or balloon dilatation, the patients were symptom-free. In three cases slight ischaemia was demonstrable during maximal exertion. Aerobic and anaerobic exercise capacity was determined by spirometry after inhalation of salbutamol (S, 0.2 mg) alone or in combination with oxitropium bromide (O, 0.2 mg). The supplementary effect of oral theophylline (T, 15 mg/kg.day) was studied in 13 patients. In terms of maximal aerobic exercise tolerance the following improvements were noted: energy output (watts): S: + 6.3%; S and O: + 12.3% ($P < 0.05$); S, O and T: + 14.0% ($P < 0.01$). Oxygen uptake (ml/min): S: + 8.2% ($P < 0.05$); S and O: + 18.2% ($P < 0.01$); S, O and T: + 35.4% ($P < 0.01$). Maximum exercise capacity was not significantly improved, although maximum oxygen uptake was significantly increased by the two-drug combination by 16.9% ($P < 0.05$) and by the three-drug combination by 19.2% ($P < 0.05$). Maximum minute volume and tidal volume rose significantly, though respiratory rate was unchanged. Heart rate and blood pressure remained practically unaffected by the treatment, both at rest and during exertion. There was no evidence of significant aggravation of ventricular arrhythmias or of ischaemia during ergometric testing.

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ACCESSION NUMBER: 0000997035 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: [Various problems of treatment of chronic prostatitis].

Nekotorye voprosy terapii bol'nykh khronicheskim
prostatitom.
AUTHOR: Kaplun, M.I. (correspondence)
SOURCE: Urologiia i nefrologiia, (1976 Sep-Oct) No. 5,
pp. 26-28.
ISSN: 0042-1154
COUNTRY: Russian Federation
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: Russian
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0000538043 EMBASE
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this record.
TITLE: [Prolonged liberation, by inclusion in an inert matrix, of
drotaverine coprecipitate].
Liberation prolongee, par inclusion dans une matrice
inerte, de la drotaverine coprecipitee..
AUTHOR: Rutz-Coudray, M.H. (correspondence); Giust, J.; Buri, P.
SOURCE: Pharmaceutica acta Helvetiae, (1979) Vol. 54, No.
12, pp. 363-365.
ISSN: 0031-6865
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: French
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0000044011 EMBASE
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this record.
TITLE: Anticholinergics in the treatment of peptic ulcer..
AUTHOR: Walan, A. (correspondence)
SOURCE: Scandinavian journal of gastroenterology. Supplement, (
1979) Vol. 55, pp. 84-95.
ISSN: 0085-5928
COUNTRY: Norway
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

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ACCESSION NUMBER: 0000028167 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of
this record.
TITLE: Urinary incontinence during treatment with depot
phenothiazines..
AUTHOR: Dainow, I.I. (correspondence)
SOURCE: British medical journal, (22 Jul 1978) Vol. 2,
No. 6132, pp. 282.
ISSN: 0007-1447
COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Letter
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010
Last Updated on STN: Mar 2010

L15 ANSWER 31 OF 31 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
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ACCESSION NUMBER: 1998:274444 BIOSIS
DOCUMENT NUMBER: PREV199800274444
TITLE: NGF delays rather than prevents the cholinergic
terminal damage and delayed neuronal death in the
hippocampus after ischemia.
AUTHOR(S): Ishimaru, Hirohisa [Reprint author]; Takahashi, Akira;
Ikarashi, Yasushi; Maruyama, Yuji
CORPORATE SOURCE: Dep. Neuropsychopharmacol., Gunma Univ. Sch. Med., 3-39-22
Showa-machi, Maebashi-shi, Gunma 371, Japan
SOURCE: Brain Research, (April 13, 1998) Vol. 789, No. 2,
pp. 194-200. print.
CODEN: BRREAP. ISSN: 0006-8993.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998

AB Cerebral ischemia induces damage of cholinergic terminals in the
hippocampus, which preceded the delayed neuronal death (DND) of the CA1
pyramidal cells. We investigated the effects of nerve growth factor (NGF)
on the cholinergic terminal damage after ischemia. Continuous
NGF infusion (0.5 mug/7 days) into the lateral ventricle before and after
5 min ischemia prevented a decrease in choline acetyltransferase
(ChAT)-immunoreactivity and disturbance of acetylcholine (ACh) release on
the 4th day after ischemia, but not on day 7, i.e., NGF infusion caused
delay in the progress of the cholinergic terminal damage. These
findings show that the cholinergic terminal damage may result
from deficiency of endogenous NGF in an ischemic brain. In addition, we
investigated whether NGF would prevent the DND after ischemia. NGF
infusion also caused delay in the progress of the DND until day 14. Our
results suggested that the neuroprotective effect of NGF on the DND may be
secondarily yielded by maintenance of communication between
cholinergic terminal and the target CA1 cell, and that prevention
of cholinergic terminal damage may be useful for the treatment
of cerebrovascular disease.

=> d his

(FILE 'HOME' ENTERED AT 10:49:58 ON 01 APR 2010)

FILE 'REGISTRY' ENTERED AT 10:50:12 ON 01 APR 2010

L1 376 S GALANTHAMINE
L2 45 S LYCORAMINE
L3 5 S RIVASTIGMINE

FILE 'CAPLUS' ENTERED AT 10:50:38 ON 01 APR 2010

L4 2481 S L1 OR L2 OR L3
L5 10 S L4 AND (DELAY?) (S) (RELEASE)
L6 10 DUP REM L5 (0 DUPLICATES REMOVED)
L7 10 S L6
L8 0 S L6 AND AD<19981123

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:46 ON 01 APR 2010

L9 4 S ACETYLCHOLINESTERASE (S) (DELAY?) (S) (RELEASE)

L10 2 DUP REM L9 (2 DUPLICATES REMOVED)
 L11 9349 S DELAY?(A)RELEASE OR PROGRAM?(A)RELEASE
 L12 1 S L11 AND ACETYLCHOLINESTERASE(S)INHIBITOR
 L13 59 S L11 AND CHOLINERGIC
 L14 54 DUP REM L13 (5 DUPLICATES REMOVED)
 L15 31 S L14 AND PD<19981123
 L16 0 S L15 AND (GALANTHAMINE OR LYCORAMINE OR RIVASTIGMINE)

=> s l11 and acetylcholine

L17 16 L11 AND ACETYLCHOLINE

=> dup rem l17

PROCESSING COMPLETED FOR L17

L18 10 DUP REM L17 (6 DUPLICATES REMOVED)

=> d l18 1-10 ibib abs

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ACCESSION NUMBER: 0016820547 EMBASE

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TITLE: Efficacy of varenicline, an alpha4beta2 nicotinic acetylcholine receptor partial agonist, vs placebo or sustained-release bupropion for smoking cessation: a randomized controlled trial..

AUTHOR: Jorenby, Douglas E (correspondence); Hays, J Taylor; Rigotti, Nancy A; Azoulay, Salomon; Watsky, Eric J; Williams, Kathryn E; Billing, Clare B; Gong, Jason; Reeves, Karen R

CORPORATE SOURCE: University of Wisconsin School of Medicine and Public Health, Center for Tobacco Research and Intervention, Madison, Wis 53711, USA.. dej@ctri.medicine.wisc.edu

SOURCE: JAMA : the journal of the American Medical Association, (5 Jul 2006) Vol. 296, No. 1, pp. 56-63.

E-ISSN: 1538-3598

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: MEDLINE

FILE SEGMENT: ClinicalTrials.gov

CLINICAL TRIAL NO.: NCT00143364

LANGUAGE: English

ENTRY DATE: Entered STN: Mar 2010

Last Updated on STN: Mar 2010

AB CONTEXT: Varenicline, a partial agonist at the alpha4beta2 nicotinic acetylcholine receptor, has the potential to aid smoking cessation by relieving nicotine withdrawal symptoms and reducing the rewarding properties of nicotine. OBJECTIVE: To determine the efficacy and safety of varenicline for smoking cessation compared with placebo or sustained-release bupropion (bupropion SR). DESIGN, SETTING, AND PARTICIPANTS: A randomized, double-blind, placebo-controlled trial conducted between June 2003 and March 2005 at 14 research centers with a 12-week treatment period and follow-up of smoking status to week 52. Of 1413 adult smokers who volunteered for the study, 1027 were enrolled; 65% of randomized participants completed the study. INTERVENTION: Varenicline titrated to 1 mg twice daily (n = 344) or bupropion SR titrated to 150 mg twice daily (n = 342) or placebo (n = 341) for 12 weeks, plus weekly brief smoking cessation counseling. MAIN OUTCOME MEASURES: Continuous abstinence from smoking during the last 4 weeks of treatment (weeks 9-12; primary end point) and through the follow-up period (weeks 9-24 and 9-52). RESULTS: During the last 4 weeks of treatment (weeks 9-12), 43.9% of participants in the varenicline group were continuously abstinent from

smoking compared with 17.6% in the placebo group (odds ratio [OR], 3.85; 95% confidence interval [CI], 2.69-5.50; P<.001) and 29.8% in the bupropion SR group (OR, 1.90; 95% CI, 1.38-2.62; P<.001). For weeks 9 through 24, 29.7% of participants in the varenicline group were continuously abstinent compared with 13.2% in the placebo group (OR, 2.83; 95% CI, 1.91-4.19; P<.001) and 20.2% in the bupropion group (OR, 1.69; 95% CI, 1.19-2.42; P = .003). For weeks 9 through 52, 23% of participants in the varenicline group were continuously abstinent compared with 10.3% in the placebo group (OR, 2.66; 95% CI, 1.72-4.11; P<.001) and 14.6% in the bupropion SR group (OR, 1.77; 95% CI, 1.19-2.63; P = .004). Treatment was discontinued due to adverse events by 10.5% of participants in the varenicline group, 12.6% in the bupropion SR group, and 7.3% in the placebo group. The most common adverse event with varenicline was nausea, which occurred in 101 participants (29.4%). CONCLUSIONS: Varenicline is an efficacious, safe, and well-tolerated smoking cessation pharmacotherapy. Varenicline's short-term and long-term efficacy exceeded that of both placebo and bupropion SR. TRIAL REGISTRATION: clinicaltrials.gov Identifier: NCT00143364.

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ACCESSION NUMBER: 0016820546 EMBASE

COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.

TITLE: Varenicline, an alpha4beta2 nicotinic acetylcholine receptor partial agonist, vs sustained-release bupropion and placebo for smoking cessation: a randomized controlled trial..

AUTHOR: Gonzales, David (correspondence); Rennard, Stephen I; Nides, Mitchell; Oncken, Cheryl; Azoulay, Salomon; Billing, Clare B; Watsky, Eric J; Gong, Jason; Williams, Kathryn E; Reeves, Karen R

CORPORATE SOURCE: Smoking Cessation Center, Department of Medicine, Oregon Health and Science University, Portland, OR 97239, USA..

SOURCE: JAMA : the journal of the American Medical Association, (5 Jul 2006) Vol. 296, No. 1, pp. 47-55.
E-ISSN: 1538-3598

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: MEDLINE

FILE SEGMENT: ClinicalTrials.gov

CLINICAL TRIAL NO.: NCT00141206

LANGUAGE: English

ENTRY DATE: Entered STN: Mar 2010

Last Updated on STN: Mar 2010

AB CONTEXT: The alpha4beta2 nicotinic acetylcholine receptors (nAChRs) are linked to the reinforcing effects of nicotine and maintaining smoking behavior. Varenicline, a novel alpha4beta2 nAChR partial agonist, may be beneficial for smoking cessation. OBJECTIVE: To assess efficacy and safety of varenicline for smoking cessation compared with sustained-release bupropion (bupropion SR) and placebo. DESIGN, SETTING, AND PARTICIPANTS: Randomized, double-blind, parallel-group, placebo- and active-treatment-controlled, phase 3 clinical trial conducted at 19 US centers from June 19, 2003, to April 22, 2005. Participants were 1025 generally healthy smokers (> or =10 cigarettes/d) with fewer than 3 months of smoking abstinence in the past year, 18 to 75 years old, recruited via advertising. INTERVENTION: Participants were randomly assigned in a 1:1:1 ratio to receive brief counseling and varenicline titrated to 1 mg twice per day (n = 352), bupropion SR titrated to 150 mg twice per day (n = 329), or placebo (n = 344) orally for 12 weeks, with 40 weeks of nondrug follow-up. MAIN OUTCOME MEASURES: Primary outcome was the exhaled carbon monoxide-confirmed 4-week rate of continuous abstinence from smoking for

weeks 9 through 12. A secondary outcome was the continuous abstinence rate for weeks 9 through 24 and weeks 9 through 52. RESULTS: For weeks 9 through 12, the 4-week continuous abstinence rates were 44.0% for varenicline vs 17.7% for placebo (odds ratio [OR], 3.85; 95% confidence interval [CI], 2.70-5.50; P<.001) and vs 29.5% for bupropion SR (OR, 1.93; 95% CI, 1.40-2.68; P<.001). Bupropion SR was also significantly more efficacious than placebo (OR, 2.00; 95% CI, 1.38-2.89; P<.001). For weeks 9 through 52, the continuous abstinence rates were 21.9% for varenicline vs 8.4% for placebo (OR, 3.09; 95% CI, 1.95-4.91; P<.001) and vs 16.1% for bupropion SR (OR, 1.46; 95% CI, 0.99-2.17; P = .057). Varenicline reduced craving and withdrawal and, for those who smoked while receiving study drug, smoking satisfaction. No sex differences in efficacy for varenicline were observed. Varenicline was safe and generally well tolerated, with study drug discontinuation rates similar to those for placebo. The most common adverse events for participants receiving active-drug treatment were nausea (98 participants receiving varenicline [28.1%]) and insomnia (72 receiving bupropion SR [21.9%]). CONCLUSION: Varenicline was significantly more efficacious than placebo for smoking cessation at all time points and significantly more efficacious than bupropion SR at the end of 12 weeks of drug treatment and at 24 weeks. TRIAL REGISTRATION: clinicaltrials.gov Identifier: NCT00141206.

L18 ANSWER 3 OF 10 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 0015543927 EMBASE

COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.

TITLE: Elevating our therapeutic expectations in overactive bladder..

AUTHOR: Sand, Peter K (correspondence)

CORPORATE SOURCE: Department of Obstetrics and Gynecology, Northwestern University Feinberg School of Medicine Evanston, Illinois, USA..

SOURCE: Journal of the American Academy of Nurse Practitioners, (Oct 2004) Vol. 16, No. 10 Suppl, pp. 8-11.

Refs: 17

ISSN: 1041-2972

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: MEDLINE

LANGUAGE: English

ENTRY DATE: Entered STN: Mar 2010

Last Updated on STN: Mar 2010

AB Drug therapy for overactive bladder (OAB) most commonly includes antimuscarinic agents, which work by relaxing bladder smooth muscle through inhibition of acetylcholine receptors in the bladder. The major adverse effects with existing antimuscarinic agents are anticholinergic in nature (e.g., dry mouth, constipation, blurred vision). Oxybutynin and tolterodine have been used for several years for treatment of OAB; both are available in immediate- and extended-release formulations. Fewer or less severe adverse effects are reported with the extended- versus the immediate-release formulations, with little or no difference in efficacy. Oxybutynin is also available as a transdermal patch. Trospium, which was recently approved for use in the United States, has efficacy and an incidence of dry mouth similar to existing agents but does not cross the blood-brain barrier. It requires twice-daily dosing. Two new antimuscarinic agents--darifenacin and solifenacin--are in development. Both show significantly better efficacy compared with placebo for key symptoms of OAB, including urgency. The incidence of dry mouth at the lowest effective dose is 19% for darifenacin and 8% and 14% for solifenacin (2 studies).

L18 ANSWER 4 OF 10 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:203457 BIOSIS

DOCUMENT NUMBER: PREV200400204000

TITLE: basal ganglia neuronal teachers: cooperating different messages.

AUTHOR(S): Morris, G. [Reprint Author]; Arkadir, D. [Reprint Author]; Nevet, A. [Reprint Author]; Bergman, H. [Reprint Author]

CORPORATE SOURCE: Physiology, Hebrew Univ, Hebrew Univ, Jerusalem, Israel

SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 704.12.
<http://sfn.scholarone.com>. e-file.
 Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.

DOCUMENT TYPE: Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 14 Apr 2004
 Last Updated on STN: 14 Apr 2004

AB Adaptive behavior requires the evaluation of environmental stimuli with respect to their behavioral significance. Dopamine and acetylcholine neurons, acting in the striatum, emit signals related to the behavioral significance of environmental events and therefore can perform such an evaluation. Furthermore, both substances affect plasticity of cortico-striatal transmission and are therefore good candidates to serve as teachers of the basal ganglia system. Finally, the most common degenerative disease of the basal ganglia, Parkinson's disease, exhibits dysfunction of both systems. We recorded from 132 SNc Dopamine neurons and 97 putamen TANs, believed to be acetylcholine interneurons, while monkeys performed a probabilistic instrumental conditioning delayed-release task. Our results suggest that the messages carried by the two systems are functionally distinct. While both neuronal populations responded to the behaviorally significant events, the dopaminergic response reflects mismatch between expectation and outcome, complying with the teacher hypothesis. By contrast, acetylcholine neurons respond indifferently to the reward predictability or value. In addition, we found that the TANs are highly temporally correlated, whereas dopamine neurons showed very low correlation values. Finally, the pause in firing of the TANs and the burst in firing of the DA neurons coincide although they occur at different latencies for different events. Hence we propose that the response of the acetylcholine neurons illuminates the timing of potentially significant events indicating for the time that requires learning ("school-bell effect"), allowing the dopaminergic neurons ("teachers") a time frame to teach the system in view of those events. The combined effect of these two cooperating teaching signals yields an appropriate alteration of the strength of cortico-striatal connections.

L18 ANSWER 5 OF 10 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2002222065 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11958521

TITLE: Dizocilpine inhibits amphetamine-induced formation of nitric oxide and amphetamine-induced release of amino acids and acetylcholine in the rat brain.

AUTHOR: Kraus Michaela M; Bashkatova Valentina; Vanin Anatoly; Philippu Athineos; Prast Helmut

CORPORATE SOURCE: Department of Pharmacology and Toxicology, University of Innsbruck, Austria.

SOURCE: Neurochemical research, (2002 Mar) Vol. 27, No. 3, pp. 229-35.
 Journal code: 7613461. ISSN: 0364-3190. L-ISSN: 0364-3190.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 18 Apr 2002
Last Updated on STN: 8 Oct 2002
Entered Medline: 4 Oct 2002

AB Glutamate receptor activation participates in mediation of neurotoxic effects in the striatum induced by the psychomotor stimulant amphetamine. The effects of the non-competitive NMDA receptor antagonist dizocilpine (MK-801) on amphetamine-induced toxicity and formation of nitric oxide (NO) in both striatum and cortex and on induced transmitter release in the nucleus accumbens were investigated. Repeated, systemic application of amphetamine elevated striatal and cortical lipid peroxidation and NO production. Moreover, amphetamine caused an immediate release of acetylcholine and aspartate and a delayed release of GABA in the nucleus accumbens. Surprisingly, glutamate release was not affected. Dizocilpine abolished the amphetamine-induced lipid peroxidation and NO production in striatum and cortex and diminished the elevation of neurotransmitter release. These findings suggest that amphetamine evokes neurotoxic effects in both striatal and cortical brain areas that are prevented by inhibiting NMDA receptor activation. The amphetamine-induced acetylcholine, aspartate and GABA release in the nucleus accumbens is also mediated through NMDA receptor-dependent mechanisms. Interestingly, the enhanced aspartate release might contribute to NMDA receptor activation in the nucleus accumbens, while glutamate does not seem to mediate amphetamine-evoked transmitter release in this striatal brain area.

L18 ANSWER 6 OF 10 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 1998:274444 BIOSIS

DOCUMENT NUMBER: PREV199800274444

TITLE: NGF delays rather than prevents the cholinergic terminal damage and delayed neuronal death in the hippocampus after ischemia.

AUTHOR(S): Ishimaru, Hirohisa [Reprint author]; Takahashi, Akira; Ikarashi, Yasushi; Maruyama, Yuji

CORPORATE SOURCE: Dep. Neuropsychopharmacol., Gunma Univ. Sch. Med., 3-39-22 Showa-machi, Maebashi-shi, Gunma 371, Japan

SOURCE: Brain Research, (April 13, 1998) Vol. 789, No. 2, pp. 194-200. print.

CODEN: BRREAP. ISSN: 0006-8993.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 24 Jun 1998

Last Updated on STN: 24 Jun 1998

AB Cerebral ischemia induces damage of cholinergic terminals in the hippocampus, which preceded the delayed neuronal death (DND) of the CA1 pyramidal cells. We investigated the effects of nerve growth factor (NGF) on the cholinergic terminal damage after ischemia. Continuous NGF infusion (0.5 mug/7 days) into the lateral ventricle before and after 5 min ischemia prevented a decrease in choline acetyltransferase (ChAT)-immunoreactivity and disturbance of acetylcholine (ACh) release on the 4th day after ischemia, but not on day 7, i.e., NGF infusion caused delay in the progress of the cholinergic terminal damage. These findings show that the cholinergic terminal damage may result from deficiency of endogenous NGF in an ischemic brain. In addition, we investigated whether NGF would prevent the DND after ischemia. NGF infusion also caused delay in the progress of the DND until day 14. Our results suggested that the neuroprotective effect of NGF on the DND may be secondarily yielded by maintenance of communication between cholinergic

terminal and the target CA1 cell, and that prevention of cholinergic terminal damage may be useful for the treatment of cerebrovascular disease.

L18 ANSWER 7 OF 10 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1991366328 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1653822
TITLE: Physiological properties of newly formed synapses between sympathetic preganglionic neurons and sympathetic ganglion neurons.
AUTHOR: Hume R I; Honig M G
CORPORATE SOURCE: Department of Biology, University of Michigan, Ann Arbor 48109.
CONTRACT NUMBER: NS21043 (United States NINDS NIH HHS)
SOURCE: Journal of neurobiology, (1991 Apr) Vol. 22, No. 3, pp. 249-62.
Journal code: 0213640. ISSN: 0022-3034. L-ISSN: 0022-3034.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 3 Nov 1991
Last Updated on STN: 3 Nov 1991
Entered Medline: 15 Oct 1991

AB We have examined the physiological properties of transmission at newly formed synapses between sympathetic preganglionic neurons and sympathetic ganglion neurons in vitro. Chick neurons were labeled with fluorescent carbocyanine dyes before they were placed into culture (Honig and Hume, 1986), and were studied by making intracellular recordings during the first 2 weeks of coculture. Evoked monosynaptic excitatory postsynaptic potentials (EPSPs) were not observed until 48 h of coculture. Beyond this time, the frequency with which connected pairs could be found did not vary greatly with time. With repetitive stimulation, the evoked monosynaptic EPSPs fluctuated in amplitude from trial to trial and showed depression at frequencies as low as 1 Hz. To gain further information about the quantitative properties of transmission at newly formed synapses, we analyzed the pattern of fluctuations of delayed release EPSPs. In mature systems, delayed release EPSPs are known to represent responses to single quanta, or to the synchronous release of a small number of quanta. For more than half of the connections we studied, the histograms of delayed release EPSPs were extremely broad. This result suggested that either quantal responses are drawn from a continuous distribution that has a large coefficient of variation or that there are several distinct size classes of quantal responses. The pattern of fluctuations of monosynaptic EPSPs was consistent with both of these possibilities, and was inconsistent with the possibility that monosynaptic EPSPs are composed of quantal subunits with very little intrinsic variation. Although variation in the size of responses to single quanta might arise in a number of ways, one attractive explanation for our results is that the density and type of acetylcholine receptors varies among the different synaptic sites on the surface of developing sympathetic ganglion neurons.

L18 ANSWER 8 OF 10 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1982247057 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6124928
TITLE: Effects of dopamine and dibutyryl cyclic adenosine monophosphate on delayed release of transmitter at the rat neuromuscular junction.

AUTHOR: Heinonen E
 SOURCE: Pflugers Archiv : European journal of physiology, (1982
 Apr) Vol. 393, No. 2, pp. 144-7.
 Journal code: 0154720. ISSN: 0031-6768. L-ISSN: 0031-6768.
 PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
 DOCUMENT TYPE: (IN VITRO)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198209
 ENTRY DATE: Entered STN: 17 Mar 1990
 Last Updated on STN: 6 Feb 1995
 Entered Medline: 10 Sep 1982

AB The effects of dopamine and dibutylryl cyclic adenosine monophosphate (db-cAMP) on delayed release of transmitter were studied in vitro in the phrenic nerve-diaphragm muscle preparation in the rat using intracellular recording techniques. Dopamine at 1×10^{-4} mol l⁻¹ prevented the initial facilitation of delayed release of transmitter. This inhibitory phase was transformed into a transient facilitation of delayed release. We observed that dopamine hyperpolarized muscle fibres by about 9%. Thus motor nerve terminals may also have been hyperpolarized by dopamine; however, it is unlikely that this hyperpolarization explains the observed effects on delayed release of transmitter. Db-cAMP at 1×10^{-3} mol l⁻¹ predominantly augmented delayed release of acetylcholine. These effects of dopamine and db-cAMP on delayed release of transmitter are discussed in terms of a modulation of calcium fluxes in the presynaptic nerve terminal.

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ACCESSION NUMBER: 0005940046 EMBASE
 COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
 TITLE: [Research on the anticholinergic action of some belladonna products with prolonged action]. Cercetarea actiunii anticolinergice a unor produse de beladona cu efect prelungit..
 AUTHOR: Winter, D. (correspondence); Sauvard, S.; Stanescu, C.; Enescu, L.
 SOURCE: Studii si cercetari de fiziologie, (1966) Vol. 11, No. 1, pp. 69-72.
 ISSN: 0039-3959
 COUNTRY: Romania
 DOCUMENT TYPE: Journal; Article
 FILE SEGMENT: MEDLINE
 LANGUAGE: Romanian
 ENTRY DATE: Entered STN: Mar 2010
 Last Updated on STN: Mar 2010

L18 ANSWER 10 OF 10 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 1966:47193 BIOSIS
 DOCUMENT NUMBER: PREV19664700047195; BA47:47195
 TITLE: The effect of temperature on the synaptic delay at the neuromuscular junction.
 AUTHOR(S): KATZ, B.; MILEDI, R.
 CORPORATE SOURCE: Univ. Coll. London, London, Engl., UK
 SOURCE: J PHYSIOL, (1965) Vol. 181, No. 3, pp. 656-670.
 DOCUMENT TYPE: Article

FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB The synaptic delay at the frog neuromuscular junction is greatly lengthened by lowering the temperature. At 20[degree] C, 0.5 msec elapses between the arrival of the nerve impulse in the axon terminal and the commencement of current flow through the muscle membrane. At 2.5[degree] C, the delay amounts to 3.5-7 msec (average Q10 approximately 3). This delay could be due to the following factors, delayed release of acetylcholine after arrival of the nerve impulse, slowness of diffusion from pre- to post-synaptic sites, delay in receptor reaction. During ionophoretic application of acetylcholine from a closely placed micro-pipette, depolarizations can be seen to arise as early as 0.2 msec after the beginning of the ionophoretic flow (at 2.5[degree]C). The effect of temperature on conduction velocity in the nerve terminals is relatively small (Q10 1.5-2). The release of the transmitter becomes dispersed in time at low temperature. Under suitable conditions, the release of transmitter quanta becomes "desynchronized" so that one can count them individually during successive nerve impulses and apply direct statistical tests to their numerical distribution. The quantal distribution is fitted by Poisson's theorem, in close agreement with earlier results which were based on analysis of amplitude variations of the end-plate potential. ABSTRACT
AUTHORS: Authors

=> d his

(FILE 'HOME' ENTERED AT 10:49:58 ON 01 APR 2010)

FILE 'REGISTRY' ENTERED AT 10:50:12 ON 01 APR 2010

L1 376 S GALANTHAMINE
L2 45 S LYCORAMINE
L3 5 S RIVASTIGMINE

FILE 'CAPLUS' ENTERED AT 10:50:38 ON 01 APR 2010

L4 2481 S L1 OR L2 OR L3
L5 10 S L4 AND (DELAY?) (S) (RELEASE)
L6 10 DUP REM L5 (0 DUPLICATES REMOVED)
L7 10 S L6
L8 0 S L6 AND AD<19981123

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:46 ON 01 APR 2010

L9 4 S ACETYLCHOLINESTERASE(S) (DELAY?) (S) (RELEASE)
L10 2 DUP REM L9 (2 DUPLICATES REMOVED)
L11 9349 S DELAY?(A)RELEASE OR PROGRAM?(A)RELEASE
L12 1 S L11 AND ACETYLCHOLINESTERASE(S) INHIBITOR
L13 59 S L11 AND CHOLINERGIC
L14 54 DUP REM L13 (5 DUPLICATES REMOVED)
L15 31 S L14 AND PD<19981123
L16 0 S L15 AND (GALANTHAMINE OR LYCORAMINE OR RIVASTIGMINE)
L17 16 S L11 AND ACETYLCHOLINE
L18 10 DUP REM L17 (6 DUPLICATES REMOVED)

=> s l11 and acetylcholinesterase

L19 7 L11 AND ACETYLCHOLINESTERASE

=> dup rem l19

PROCESSING COMPLETED FOR L19

L20 4 DUP REM L19 (3 DUPLICATES REMOVED)

=> d 120 1-4 ibib abs

L20 ANSWER 1 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1996407511 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8811564
TITLE: Neuropathy and vasculopathy in colonic strictures from
children with cystic fibrosis.
AUTHOR: Collins M H; Azzarelli B; West K W; Chong S K; Maguiness K
M; Stevens J C
CORPORATE SOURCE: Division of Pediatric Pathology, Indiana University School
of Medicine, Riley Hospital for Children, Indianapolis,
USA.
SOURCE: Journal of pediatric surgery, (1996 Jul) Vol. 31, No. 7,
pp. 945-50.
Journal code: 0052631. ISSN: 0022-3468. L-ISSN: 0022-3468.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 4 Dec 1996

AB Colonic strictures are rare in patients who have cystic fibrosis, but recently have developed in those who have been treated with delayed-release high-dose pancreatic enzyme supplements. Colonic strictures from eight such pediatric patients showed neural abnormalities consisting of ganglion cell hyperplasia and ectopia, and intermyenteric plexus hyperplasia. Cholinergic and adrenergic stains of mucosal nerve fibers were more prominent in histological sections of the cystic fibrosis strictures than in sections from colons of children without cystic fibrosis. The mean grade of staining with acetylcholinesterase in the lamina propria of the strictured cystic fibrosis colons was 2.38 +/- 1.25, compared with .93 +/- .93 (P < .055) in bowels from children without cystic fibrosis. The mean grade for tyrosine hydroxylase staining in the lamina propria was 2 +/- .97 in the strictures and was .79 +/- .81 (P < .05) in the bowels of children who did not have cystic fibrosis. Vasoactive intestinal peptide staining in bowels from children with cystic fibrosis with and without stricture did not differ significantly from that of children without cystic fibrosis. Vasculopathy consisting of fibrointimal hyperplasia in submucosal veins and mesenteric arteries was found only in colonic strictures owing to cystic fibrosis. Colonic strictures in patients with cystic fibrosis who received high-dose pancreatic enzyme supplements contain ganglion cell abnormalities, and mucosal cholinergic and adrenergic activity may be increased in these strictures. The stricture vasculopathy may be drug-related and/or related to increased catecholamine activity.

L20 ANSWER 2 OF 4 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 0002501967 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: Growth hormone response to overnight growth hormone-releasing hormone infusion and oral pyridostigmine in children with short stature..
AUTHOR: Ross, R.J. (correspondence); Savage, M.O.; Kirk, J.M.; Besser, G.M.
CORPORATE SOURCE: Department of Endocrinology, St Bartholomew's Hospital, London, UK..
SOURCE: Acta paediatrica Scandinavica. Supplement, (1989) Vol. 349,

pp. 114-116; discussion 123-124.

ISSN: 0300-8843

COUNTRY: Sweden
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered STN: Mar 2010

Last Updated on STN: Mar 2010

AB The development of a long-acting or depot preparation of growth hormone-releasing hormone (GHRH) may have many advantages over conventional treatment (with GH) of GH-deficient children. Pyridostigmine, an acetylcholinesterase inhibitor, has been shown to augment basal GH secretion and the GH response to GHRH in short children. It may thus provide adjuvant therapy to depot GHRH. The GH response to a nocturnal subcutaneous infusion of GHRH (1-29)NH₂ in doses of 5 and 10 micrograms/kg/hour was investigated in five short, slowly growing children. The effect of oral pyridostigmine 60 mg on nocturnal GH secretion and the GH response to a nocturnal infusion was also examined. The subcutaneous infusion of GHRH augmented pulsatile GH release in all five children. There was a dose-related response to subcutaneous GHRH for the GH area under the curve and mean GH pulse amplitude, but no change in the number of pulses. There was a significant rise in the mean baseline GH concentration during the GHRH infusion compared with placebo. Pyridostigmine had no effect on either basal or stimulated GH secretion.

L20 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1988043506 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3672538
TITLE: Acute tabun toxicity; biochemical and histochemical consequences in brain and skeletal muscles of rat.
AUTHOR: Gupta R C; Patterson G T; Dettbarn W D
CORPORATE SOURCE: Department of Pharmacology, School of Medicine, Vanderbilt University, Nashville, TN 37232.
SOURCE: Toxicology, (1987 Nov) Vol. 46, No. 3, pp. 329-41.
Journal code: 0361055. ISSN: 0300-483X. L-ISSN: 0300-483X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198712
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 17 Dec 1987

AB Male Sprague-Dawley rats injected s.c. with an acute non-lethal dose (200 micrograms/kg) of ethyl N,N-dimethylphosphoramidocyanidate (tabun) showed onset of hypercholinergic activity within 10-15 min. The maximal severity of toxicity signs was evident within 0.5-1 h and persisted for 6 h. Except for mild tremors no overt toxicity signs were evident after 24 h. Within 1 h a dramatic decline of acetylcholinesterase (AChE) activity occurred in all the brain structures (less than 3%) and skeletal muscles (less than 10% in soleus and hemi-diaphragm; and 32% in extensor digitorum longus (EDL)). No significant recovery was seen up to 48-72 h. Within 7 days rats became free of toxicity signs and AChE activity had recovered to about 40% in brain structures (except cortex, 14%) and 65-70% in skeletal muscles. Within 1 h the 16 S molecular form of AChE located at the neuromuscular junction was most severely inhibited in soleus, followed by hemi-diaphragm and least in the EDL, and had fully recovered in all the muscles when examined after day 7. Muscle fiber necrosis developed within 1-3 h in soleus and hemi-diaphragm and after a delay of 24 h in EDL. The highest number of necrotic lesions in all muscles was seen at 72 h with the hemi-diaphragm maximally affected and EDL the least.

To determine detoxification of tabun by non-specific binding, the activity of butyrylcholinesterase (BuChE) and carboxylesterase (CarbE) was measured. The inhibition and recovery pattern of BuChE activity was quite similar to that of AChE, except that the rate of recovery was more rapid. Within 1 h the remaining activity of CarbE was 10% in plasma, about 30% in brain structures, and 79% in liver; recovery was complete within 7 days. The inhibition of BuChE and CarbE can serve as a protective mechanism against tabun toxicity by reducing the amount available for AChE inhibition. The prolonged AChE inhibition in muscle and brain may indicate storage of tabun and delayed release from non-enzymic sites. Since tabun is a cyanophosphorus compound, the toxic effects from the released cyanide (CN) could be another reason for the delayed recovery after tabun.

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TITLE: [Effect of (L-Phe7) and (D-Phe7) ACTH 4-7 and an ACTH 4-7 analog with prolonged action on acetylcholinesterase activity in the rat brain].
Vliianie (L-Phe7) i (D-Phe7) AKTG 4-7 i analoga AKTG 4-7 prolongirovannogo deistviia na aktivnost' atsetilkholinesterazy golovnogo mozga krys..

AUTHOR: Aleksidze, N.G. (correspondence); Balavadze, M.V.; Ponomareva-Stepnaia, M.A.; Nezavibat'ko, V.N.; Alferova, L.I.

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AB ACTH4-7 and its long-acting analog stimulate acetylcholinesterase activity of different areas of the rat brain. Based on the data concerning the effect of an amino acid mixture equivalent to ACTH4-7 and actinomycin D on acetylcholinesterase activity of the white substance of the large hemispheres it is inferred that the oligopeptide-induced increase in the enzyme activity is linked with the induction of the synthesis of new acetylcholinesterase molecules.

=> d his

(FILE 'HOME' ENTERED AT 10:49:58 ON 01 APR 2010)

FILE 'REGISTRY' ENTERED AT 10:50:12 ON 01 APR 2010

L1 376 S GALANTHAMINE

L2 45 S LYCORAMINE

L3 5 S RIVASTIGMINE

FILE 'CAPLUS' ENTERED AT 10:50:38 ON 01 APR 2010

L4 2481 S L1 OR L2 OR L3

L5 10 S L4 AND (DELAY?) (S) (RELEASE)

L6 10 DUP REM L5 (0 DUPLICATES REMOVED)

L7 10 S L6

L8 0 S L6 AND AD<19981123

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 10:52:46 ON 01 APR 2010

L9 4 S ACETYLCHOLINESTERASE(S) (DELAY?) (S) (RELEASE)
L10 2 DUP REM L9 (2 DUPLICATES REMOVED)
L11 9349 S DELAY?(A)RELEASE OR PROGRAM?(A)RELEASE
L12 1 S L11 AND ACETYLCHOLINESTERASE(S) INHIBITOR
L13 59 S L11 AND CHOLINERGIC
L14 54 DUP REM L13 (5 DUPLICATES REMOVED)
L15 31 S L14 AND PD<19981123
L16 0 S L15 AND (GALANTHAMINE OR LYCORAMINE OR RIVASTIGMINE)
L17 16 S L11 AND ACETYLCHOLINE
L18 10 DUP REM L17 (6 DUPLICATES REMOVED)
L19 7 S L11 AND ACETYLCHOLINESTERASE
L20 4 DUP REM L19 (3 DUPLICATES REMOVED)

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

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